Fulminant Hepatic Failure (FHF) Rapid development of severe acute liver injury with impaired synthetic function and encephalopathy in person with previously normal liver or well compensated liver disease. Time course: either encephalopathy w/in 8 weeks of onset of symptoms in patient with normal liver or within 2 weeks of the development of jaundice with normal liver or previous liver disease. O/w “subfulminant”

Etiologies of FHF: “ABCDEF”
- **A**: Acetominophen, Hep A, Autoimmune hepatitis
- **B**: Hep B
- **C**: Hep C (rarely), cryptogenic
- **D**: Hep D, drugs (idiosyncratic, usually)
- **E**: Esoteric -> Wilson’s, Budd Chiari
- **F**: Fatty infiltration-> AFLP (acute fatty liver of pregnancy- seen in second half of pregnancy, usually close to term, but can be diagnoses post partum), Reyes Syndrome (associated with ASA use in children with influenza or varicella)

**Epidemiology:** Study of pts at UCSF with FHF between 1989-92: 15% Hep B, 38% Hep non A/B/C, 18% acetaminophen. Cause identified in general in about 60-80% of cases

**Hepatitis viruses:**
- **Hep A**: Most common acute viral hepatitis, but uncommonly progresses to FHF.
- **Hep B**: Precore mutant (No HepBSag or HepBeAg) – hard to diagnosis with routine serology, may be underestimated as etiology of FHF. Often mistaken as non A/B/C
- **Hep E**: Causes FHF more frequently in pregnant women in endemic areas (Asia, Africa, Middle East, Central America). Usually self limited enterically transmitted acute hepatitis, similar to Hep A. Hep E Ag tests only available for research purposes
- **Other viruses**: EBV, CMV, HSV, VZV

**Toxins:**
- **Tylenol**: Most common toxin assoc with FHF. Effect augmented with concurrent ETOh use and use of anticonvulsants
- **Amanita phalloides**
- **Anticonvulsants**: Hypersensitivity- Phenytoin, VPA
- **Halothane, Carbon Tetrachloride**: Also hypersensitivity
- **Antibiotics**: due to aberrant metabolism or hypersensitivity. INH, Ketoconazole, Rifampin, Sulfonamides, Tetracycline
- **Amiodarone, PTU, Tricyclics**
Vascular
• Portal vein thrombosis, Budd Chiari (hepatic vein thrombosis), veno-occlusive
disease, ischemic hepatitis (shock liver)

Miscellaneous
• Infiltratration of the liver with malignancy (esp lymphoma), sepsis, autoimmune
hepatitis, heat stroke

Complications of FHF: Cerebral edema, hepatorenal, hypoglycemia, metabolic
acidosis, sepsis, coagulopathy.

Prognosis: Best prognostic factors:
⇒ degree of encephalopathy (I – mild to IV- coma). Spontaneous recovery with grade I-
II ~65-70%, grade III 40-50%, grade IV <20%
⇒ Age >40 or < 10 poorer prognosis
⇒ Cause of FHF: Hep A & B, acetominophen toxicity all have better prognosis than
idiosyncratic drug reaction and Wilson’s

Kings college Criteria for OLT:
• If tylenol induced: pH<7.3 (irrespective of grade of enceph) OR grade III or IV AND
PT>100 AND Cr>3.4
• If NOT tylenol induced: PT>100 OR Any three of the following:
1) age<10 or >40,
2) etiology non A/B hep, halothane or idiosyncratic drug rxn,
3) duration of jaundice before enceph >7 days
4) PT>50
5) Bili>18